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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Cardiovascular Imaging* author instructions page.

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Patients Recovered From COVID-19 Show Ongoing Subclinical Myocarditis as Revealed by Cardiac Magnetic Resonance Imaging

The cardiovascular complications of coronavirus disease-2019 (COVID-19) are still being established (1). Expert guidelines recommend the use of cardiac imaging in the management of patients with COVID-19 (2), and cardiac magnetic resonance (CMR) has shown utility in the noninvasive detection of myocardial inflammation (3). We present a case series of 16 patients who recovered from COVID-19 who underwent CMR to assess for evidence of myocardial involvement or ongoing myocarditis.

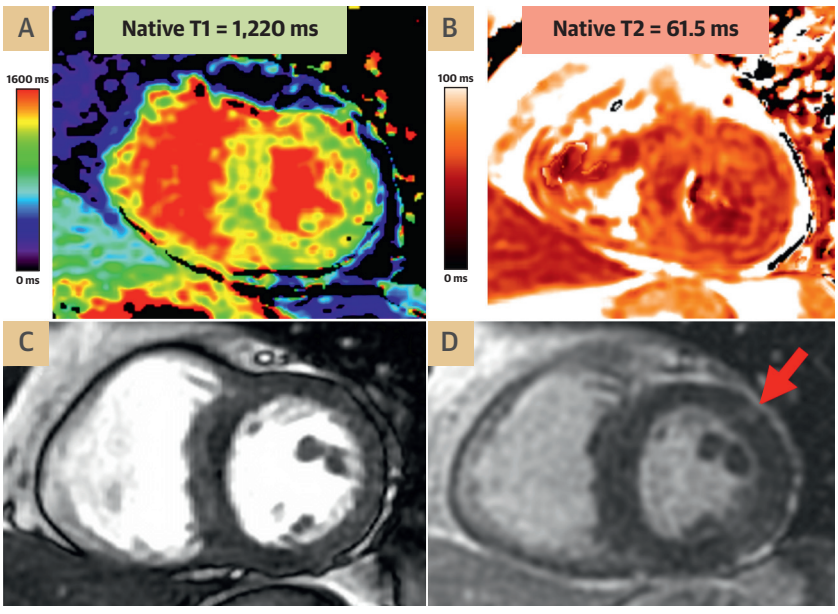
Ethics approval was obtained from the Hong Kong West Cluster (UW20-359) Institutional Review Board for this retrospective study. Inclusion criteria were COVID-19 patients admitted as inpatients to Queen Mary Hospital, referred for outpatient CMR post-recovery for raised troponin levels or electrocardiogram changes during the acute illness. Exclusion criteria were poor-quality CMR preventing assessment of ventricular function and late gadolinium enhancement (LGE). COVID-19 was diagnosed based on reverse transcription polymerase chain reaction test results of nasopharyngeal and throat swabs. Recovered COVID-19 status was based on: 1) 2 negative nasopharyngeal swab reverse transcription polymerase chain reaction results >24 h apart; and 2) absence of fever and improvement in respiratory symptoms. COVID-19 disease severity was defined according to World Health Organization criteria (4). CMR performed at 1.5-T (GE Healthcare Systems, Chicago, Illinois) included cine, native T1-mapping ($SMART_1$), T2-mapping, and LGE. T1/T2-mapping were analyzed in the mid-ventricular slice for an average value per patient. Images were reviewed independently by 3 cardiac radiologists.

Sixteen patients were identified (median age 68 years [interquartile range: 53 to 69 years]; 7 female subjects). Fifteen (94%) of the 16 patients had mild/moderate World Health Organization–defined disease severity. On admission, 14 (88%) had electrocardiogram changes, and 7 (44%) had raised troponin levels. At ≥2 weeks' post-discharge, 11 (69%) patients were asymptomatic. Five (31%) had symptoms such as cough, shortness of breath, and mild chest pain.

CMR was performed at a median of 56 days' post-recovery. Three (19%) patients had nonischemic LGE with elevated global T2-mapping values (57 to 62 ms), fulfilling the Lake Louise criteria for myocardial inflammation (3): 1 had chest discomfort with mildly elevated C-reactive protein (CRP) levels; 1 was asymptomatic but with elevated troponin levels (**Figure 1**); and 1 was asymptomatic with no blood biomarkers of inflammation. The fourth patient with LGE had a known history of non-ST-segment elevation myocardial infarction with circumflex artery stenting, showing a lateral wall infarct but no myocarditic changes. In the remainder (all 12 without LGE), 4 patients had elevated T1 only, 1 had elevated T2 only, and 1 had both elevated T1 and T2. Of these, 4 of 6 had blood biomarkers of inflammation (high white blood cell count, CRP, or troponin), and 3 of 6 had ongoing symptoms (1 cough, 1 cough/shortness of breath, and 1 shortness of breath/chest discomfort). The remaining 6 had normal T1 and T2 and no LGE; 5 of 6 were asymptomatic. Two of these 5 patients still had elevated troponin levels, 1 of 5 had elevated CRP levels, and 2 of 5 had normal blood test results. None had pericardial thickening or effusion.

Our study describes subclinical ongoing or resolving myocardial inflammation in patients recovered from COVID-19, as revealed by CMR. A study from Wuhan, China, reported that 58% of patients who recovered from COVID-19 had abnormal CMR findings but all had cardiac symptoms (5). In contrast, our study extends that although 69% (11 of 16) of patients who recovered from COVID-19 were asymptomatic, a majority (56% [9 of 16]) exhibited abnormal CMR findings (high T1 and/or T2, ± nonischemic LGE), 67% (6 of 9) of whom had accompanying blood biomarkers of ongoing inflammation, even if asymptomatic (3 of 6). In asymptomatic patients, 45% (5 of 11) had abnormal CMR findings; 27% (3 of 11) of asymptomatic patients also had

FIGURE 1 **CMR Images From a Recovered Asymptomatic COVID-19 Patient With Myocardial Inflammation**



E	Cardiac Magnetic Resonance (CMR)
LV End-Diastolic Volume Indexed (ml/m^2)	79 (IQR: 70-84)
LV Ejection Fraction (%)	59 (IQR 56-65)
RV End-Diastolic Volume Indexed (ml/m^2)	88 (IQR 76-94)¶
RV Ejection Fraction (%)	53 (IQR 48-57)
Global mid-ventricular native T1 (ms)	1,209 (IQR 1,164-1,219)#
Global mid-ventricular native T2 (ms)	52 (IQR 50-56)##
High global native T1 only (>1,208 ms), n (%)	4 (25%)
High global native T2 only (>54.8 ms), n (%)	1 (5%)
High native T1 and T2, n (%)	4 (25%)

(A and B) High global T1- and T2-mapping values. **(C)** Short-axis cine. **(D)** Small, subepicardial, basal anterolateral wall late gadolinium enhancement **(arrow)**. **(E)** Cardiac magnetic resonance (CMR) results. ¶ = 1 patient had borderline dilated right ventricle (RV) and dilated main pulmonary artery (37 mm), with no initial suspicion of pulmonary embolus, and a ventilation/perfusion scan post-CMR was normal. #p < 0.02 when compared to 15 healthy volunteers with a mean T1 of 1,158 25 ms (2SD range 1,190 to 1,208 ms). ##p < 0.01 when compared to 15 healthy volunteers with a mean T2 of 48.2 ± 3.4 ms (2SD range 41.5 to 54.8 ms). COVID-19 = coronavirus disease-2019; IQR = interquartile range; LV = left ventricular; V/Q = ventilation/perfusion.

corroborating serological evidence of inflammation. In symptomatic patients, 80% (4 of 5) had abnormal CMR findings (high T1 and/or T2), 75% (3 of 4) of whom had corroborating serological evidence of ongoing inflammation. Overall, 6 of 16 (38%) patients had both imaging and serological evidence of myocardial inflammation, and may need follow-up within their individual clinical context. Three (19%) patients had either high T1 and/or T2 on CMR but without blood biomarkers of inflammation; the abnormal T1 or T2 signals may represent residual or resolving myocardial inflammation. Thus, in patients with COVID-19 deemed to have recovered, there remains a high index of suspicion of initial and ongoing myocardial inflammation, and CMR has demonstrable utility in identifying subclinical myocardial involvement post-COVID-19.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Cardiovascular Imaging* author instructions page.

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Frequency of Pulmonary Embolism in Patients With COVID-19

Several reports have highlighted a high incidence of pulmonary embolism (PE) in patients with coronavirus disease-2019 (COVID-19) (1–4). However, patients with severe illness, particularly those in the intensive care unit, have increased D-dimer levels and high risk of venous thromboembolism. It is not clear whether the high risk of PE reported in some series simply reflects severe illness, or whether COVID-19 itself confers a particularly high risk of thromboembolism. We thus set out to evaluate the rate of PE in patients with COVID-19 compared with control subjects who were tested but found to be negative for the virus.

This retrospective study was approved by the Institutional Review Board of our hospital system. Patients in our health care system who received COVID-19 reverse transcriptase polymerase chain reaction testing between March 1, 2020, and May 1, 2020, and underwent computed tomography pulmonary angiography (CTPA) within 7 days prior to and 14 days after the testing were included. Radiology reports and D-dimer levels within 1 day of the CTPA were retrieved from the electronic medical record. Clinical pre-test probability of PE was derived from order indications. The presence of an endotracheal tube was extracted from radiology reports. CTPA reports were recorded as positive for PE, negative, or nondiagnostic. Nondiagnostic studies were excluded.

Statistical analysis was performed with JMP Pro version 15 (SAS Institute, Cary, North Carolina). Differences between categorical variables were analyzed with 1-tailed Fisher exact test and continuous variables with 1-tailed Student's *t*-test. Multivariable logistic regression was used to evaluate determinants of a positive CTPA. Confidence intervals (CIs) for area under the receiver-operating characteristic curves were generated by bootstrapping.

A total of 709 CTPAs were identified; 13 nondiagnostic studies were excluded, leaving 696 CTPAs from 674 patients. The median age was 63 (range 17 to 100) years, 352 patients (52%) were female, and 167 (25%) were COVID-19-positive. Pre-test probability was available for 475 CTPAs (68%): of COVID-19-negative patients, 33 (9%) were low risk, 152 (43%) intermediate risk, and 169 (48%) high risk; of COVID-19-positive patients, 6 (5%) were low risk, 74 (61%) intermediate risk, and 41 (34%) high risk. The 167 patients with COVID-19 represent 3% of the total number (n = 5,816) of patients who were COVID-19-positive in our health care system in that time frame.

Of 170 CTPAs in patients with COVID-19, 20 (11.8%) were positive for PE, compared with 45 of 526 CTPAs (8.6%) in patients without COVID-19 (p = 0.14) (**Table 1**). Of patients intubated at the time of CTPA, 7 of 23 patients with COVID-19 (30.4%) had PE, compared with 6 of 33 patients without COVID-19 (18.2%) (p = 0.22). For CTPAs done within 1 day of COVID-19 testing, 14 of 99 patients with COVID-19 (14.1%) had PE versus 29 of 375 patients without COVID-19 (7.7%) (p = 0.04). PEs were located in the main pulmonary arteries in 11 patients (17%), lobar in